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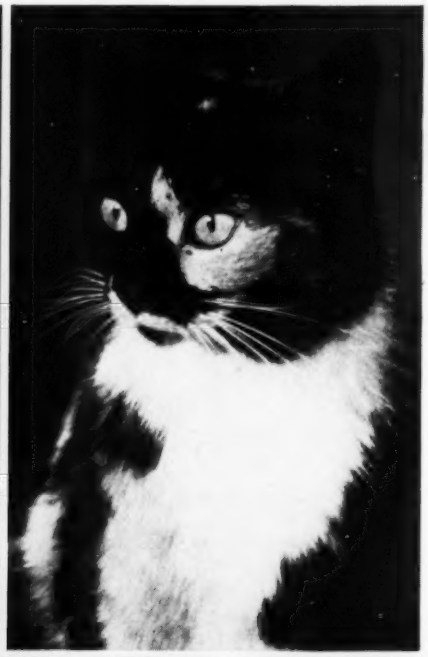

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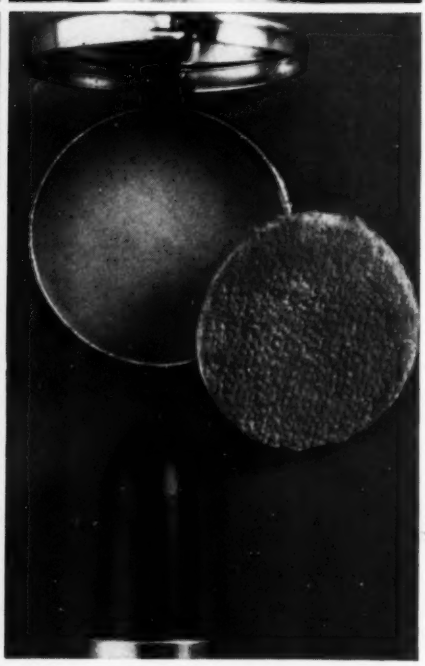

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Transfusion Reactions

Chairman: Harry D. Ellis, M.D.
Seminar Summary: E. Eric Muirhead, M.D.

St. Vincent Hospital, Santa Fe
February 28, 1961

Case Preparations: H. R. Landmann, M.D.

Case No. 1: This 38 year old housewife was admitted to St. Vincent Hospital at 11:00 a.m. 1-12-53, with a chief complaint of vaginal bleeding. The bleeding was described as profuse and of five days duration. Examination revealed extreme pallor, blood pressure 88/40 mm Hg., and a rapid pulse. The hemoglobin level was 7.0 grams/100 ml.

At 1:00 p.m. placental tissue was removed from the uterus by curettage. Two units of blood were ordered for the immediate post-operative period. At 11:15 p.m. 500 cc. of blood were started. The patient became nauseated, vomited, and began having chills. She became cyanotic and the blood pressure was found to be 80/30 mm Hg.

Blood Discontinued

The attending physician was notified and he ordered the blood discontinued. The amount of blood given was not recorded. Oxygen was started and Coramine was given. Over the next three hours the patient improved clinically and her blood pressure rose to 100/70. The next morning catheterization yielded only 15 cc. of dark urine.

Investigation of the blood given the night before revealed that the blood taken from the blood bank had not been typed nor had it been crossmatched. The urine output for the next seven days ranged from 30 to 100 cc. each day. Fluid intake was restricted. The blood urea nitrogen rose to 55 mg. per 100 ml. On 1-21-53, the urine output improved to 400 cc. and subsequently increased to normal. The patient was discharged in good condition on 1-30-53.

Program supported by a grant-in-aid from Merck, Sharp & Dohme postgraduate program.

Case No. 2: On 5-17-59, at age five weeks, this baby was found to have a hemoglobin of 2.0 grams. Bone marrow examination revealed an absence of nucleated red cells and normal myelopoiesis. Additional studies by Dr. Maxwell Wintrobe in Salt Lake City confirmed the diagnosis of congenital red cell aplasia.

From birth to the present time, various therapeutic efforts have failed to stimulate erythropoiesis and the baby requires periodic transfusions. During the first eight months of life, no transfusion reactions were noted. On 1-23-60 and 1-24-60, temperature elevations of 102° and 103° were noted, following each of 250 ml. infusions of blood.

Generalized Urticaria

Transfusions without reaction were given on 5-14-60, 6-25-60 and 8-21-60. Following a transfusion on 11-23-60, the baby developed generalized urticaria.

On 12-22-60, the baby again developed generalized urticaria and also edema of the feet following transfusion despite prior administration of Benadryl. On 12-23-60 the second half of the unit given 12-22-60 was administered following administration of Benadryl and Medrol. The plasma was removed from this second half unit and Medrol was also given during the infusion. No reaction occurred and the hemoglobin response was good.

On 2-20-61 the baby was transfused without reaction with blood from which the plasma had been removed.

Case No. 3: This 42 year old white female patient with a previous duodenal ulcer entered the hospital with tarry stools and a moderate anemia.

A subtotal gastrectomy was performed on 9-3-54, following which the patient complained of "red vision".

Hemorrhages were found in the fundi, leading to a diagnosis of aplastic anemia on the basis of an aplastic bone marrow revealed by aspiration and operative marrow biopsy. In March of 1955, the patient was seen by Dr. Maxwell Wintrobe in Salt Lake City who confirmed the diagnosis but requested a return for further marrow study.

Severe Reaction

At this time, her ninth blood transfusion resulted in a severe febrile reaction lasting almost eight hours. Her temperature rose to 104° and she suffered severe aching and chills. Subsequent transfusions were associated with mild reactions although Benadryl was given parenterally before and after transfusion.

In April, 1955, a repeat marrow study by Dr. Maxwell Wintrobe led to a diagnosis of monocytic leukemia. An unknown number of transfusions were given at this time in Salt Lake City, following which Dr. Wintrobe suggested warming the blood to body temperature prior to transfusion because of the development of cold agglutinins in the patient. In the ensuing 14 months the patient received two to three transfusions per month with the blood given at body temperature. There were only five moderately severe febrile reactions and one reaction associated with mild urticaria. At no time did the patient become jaundiced nor was the urine discolored.

Blood Warmed

After April 1955, all transfusions were given with blood warmed to 37° C. and the tubing placed in a 37°C. water bath. In addition the patient received Benadryl 50 mgm. orally four times a day during the course of each hospitalization. In all, exclusive of blood given in Salt Lake City, the patient received 61 transfusions at this hospital.

The first eight were without reactions. The second 13 were associated with moderate to severe febrile reactions. The remaining 40 given with the blood warmed to body temperature were associated with five moderately severe febrile reactions, one reaction with mild urticaria, and twelve with mild febrile reactions. During her final two admissions, the patient received nine units of blood without reaction.

Discussion and Treatment: Richard Angle, M.D.:

In discussing the three cases presented tonight it seems logical to first outline briefly the type of transfusion reactions. It is of interest to note that Miale reports the incidence of transfusion reactions in general to be about 5 per cent and he further states that an incidence below this figure means inadequate reporting of reactions, whereas an incidence much above this figure indicates the need for a thorough review of the entire transfusion service.

Most Common Reaction

The most common reaction is the "chill-fever reaction" caused by impurities in solutions or equipment and also by agglutinins against leukocytes and platelets as well as certain components in plasma. Allergic reactions are the second most common reactions and are manifested by hives, asthma, or rarely anaphylaxis.

The hemolytic reaction is the most dangerous and results from true incompatibility of blood types. This leads to serious and sometimes fatal renal tubular necrosis.

Hemoclastic Reactions

Less common reactions are "hemoclastic" reactions due to some unidentified component of plasma, hemorrhage due to possible post-transfusion thrombocytopenia, circulatory overloading due to excess volume or excess speed resulting in cardiac failure, and citric acid intoxication in massive transfusions.

Of the cases presented tonight, Case No. 1 represents a typical hemolytic reaction due to incompatible blood. Unfortunately the amount of blood given was not recorded but it was likely not great inasmuch as complete anuria did not develop and recovery was complete in 18 days.

Case No. 2 illustrates two types of reactions. One was pyrogenic, apparently due to some plasma component or leukoagglutinin with sensitization developing over an eight month period. In addition this child had two allergic reactions manifested by urticaria. Both reactions were benefitted by removal of plasma from the donor blood, and antihistamines, and cortico-steroids. Also some of the buffy coat may have been removed along with the plasma.

Case No. 3, with whom I had to contend in life, manifested pyrogenic reactions, due in large part to cold agglutinins which sometimes develop after many transfusions.

Warming the donor blood to body temperature and running the tubing through a 37° water bath materially reduced the severity of the reactions.

I suspect that this patient may also have developed leukoagglutinins which contributed some part of her reaction. In addition one mild allergic reaction was noted.

It seems, therefore that the cases illustrate the three most common transfusion reactions, hemolytic, allergic, and pyrogenic.

Treatment

Little can be said about the treatment of reactions. Obviously the best treatment is prevention. Faced with an allergic reaction, parenteral epinephrine and antihistamines are indicated and effective. The treatment of a hemolytic reaction is to stop the blood as soon as a reaction is known or suspected and to institute expectant treatment for lower nephron nephrosis.

In this regard the immediate alkalinization of the patient with Bicarbonate has been advocated.

This has been said to help prevent deposition of heme pigments in the renal tubules. Since it is apparently not the pigments but renal ischemia possibly due to shock that actually produces the damage, and since it does not seem wise to load sodium into the patient with impending renal failure, I believe this treatment is highly questionable.

It would be wiser to overcome shock with compatible blood, plasma, or norepinephrine. Customary treatment of renal shutdown is then indicated.

For the pyrogenic reaction, Aspirin gr. X may shorten the course as will also calcium gluconate 10 ml. intravenously particularly when given along with Morphine Sulfate gr. ¼.

We will have more to say about treatment as the discussion progresses.

Dr. Ellis: Our next speaker will be Dr. Eric Muirhead. Dr. Muirhead is Professor of Clinical Pathology at Wayne State University School of Medicine and is head of the Pathology Department of Woman's Hospital, Detroit, Michigan. We are very fortunate in having Dr. Muirhead here this evening.

Etiology: E. Eric Muirhead, M.D.:

We spent some time on this subject this afternoon and the speakers have covered some aspects of it already.

What I would like to do now is to single out one or two features that have to do particularly with the cases presented.

With respect to the first case, it obviously appears to be one of an incompatible transfusion reaction followed by a degree of acute renal failure. A term, somewhat more popular now, applied to this condition is "Acute Tubular Necrosis".

This case brings forth questions concerning the background of hemolysis and perhaps the relationship of hemolysis to renal failure.

In regard to hemolysis, one could suggest Mollison's definition of an incompatible transfusion as the most practical one from a clinical standpoint. He states that by an incompatible transfusion, one means that there is a shortening of the life span of the red cells due to an **in vivo** reaction between antibodies and red cells.

The antibodies most often are circulating in the recipient, and act against the donor's red cells. This same definition would apply when a high titre of antibody is transfused in plasma and the antibody of the donor reacts with the recipient's red cells.

There are several known aspects of this reaction which play on the magnitude of the destruction of red cells. I think it is fair to say that there is every indication that the rate of destruction, as well as the number of red cells destroyed, has a bearing on what happens to the individual clinically.

The rate of destruction of the red cells may be graded in terms of the adversity to the recipient, so that if the red cells of 500 cc. of blood are destroyed in a few minutes, then the recipient develops a most severe degree of abnormality, not only renal failure, but a hemorrhagic diathesis. From this, the reactions are graded downward to the most mild type of reaction.

This is why we like to separate incompatible reactions under those with hemoglobinuria and those without hemoglobinuria because those with hemoglobinuria have a more violent, a more rapid, and a substantially greater destruction of

red cells and tend to be associated with renal failure, whereas those without hemoglobinuria have a lesser degree of destruction and tend not to be associated with renal failure. In most cases, one can separate those with and those without renal failure by this means.

One would have expected the first case presented to have had hemoglobinuria and certainly hemoglobinemia in view of the subsequent development of renal failure.

Type of Antigen

Among the factors which play a part in the determination of the amount of hemolysis is included the type of antigen, though this is often not a prominent factor. Where this is particularly pertinent is in the transfusion of A1 and A2 red cells. If one transfuses a subject with type A1 red blood cells, with a blood containing anti-A antibodies, the rate of destruction of cells will be greater than if the recipient were type A2. This of course would have a bearing on whether there will be hemoglobinuria and renal failure.

The number of red cells transfused has a bearing because the numbers that are destroyed are related to the numbers that are transfused. If a lesser volume is transfused, then the volume of the destroyed red cells will be minimized, the adversity therefore minimized. Of particular importance is the titer of the antibodies involved in the reaction and the type of the antibodies. With a low titre, one tends to have lesser degree of destruction, lesser adversity and these incompatibilities may cause only a chill-fever reaction without hemoglobinuria.

The type of antibody has a bearing because certain antibodies are more violent and destructive to red cells than others. Antibodies that fix complement or depend on complement are more violently hemolytic than those that do not depend on complement, and therefore the rate of destruction may be related to the type of antibody. Hemolysins are the most destructive antibodies, however nonhemolysins may be quite destructive.

Therefore, in considering an incompatible type of transfusion, one should keep in mind the various factors that enter into the rate of destruction of the red blood cells. It is quite obvious that whatever the incompatibility was in case No. 1, the rate of destruction was substantial and enough to be related to acute renal failure.

Renal Failure

It does not necessarily follow that a person receiving incompatible blood will develop renal failure. There are good examples, we have had some ourselves, where a substantial transfusion though incompatible, was not followed by renal failure. Most of the hemoglobinuric forms are followed by renal failure. I think that one can summarize this by saying that circulatory failure affects the development of acute tubular necrosis, and if one has peripheral circulatory failure plus hemoglobinuria, then the likelihood of acute tubular necrosis or acute renal failure will be accentuated.

In respect to the second case, I will have to curtail my comments because it is a pediatric case, and I have dealt in my experience mostly with adults. I would like to reflect upon the problem presented by this case as it would pertain to an adult.

I will increase this youngster's age to about 35, and discuss it accordingly. These aregenerative or aplastic anemias require many transfusions, and may in time be associated with splenomegaly, and eventually the question arises as to whether a shortened life span of the transfused cells bears a relationship to the splenomegaly.

Demands Lessened

The cells often do not remain in the circulation as long as expected, and whereas this has no bearing on a transfusion reaction itself, it has a bearing on the transfusion demand. It has been my impression that if one removes the spleen under these conditions, the demands for transfusion may be lessened but it appears to be lessened not because the spleen itself is doing something specifically in a hemolytic sense, but because the life span of the transfused cells are temporarily prolonged. This is by virtue of the fact that the large spleen forms a graveyard for cells which were likely subjected to a form of incompatibility. As time goes by, this effect of splenectomy may be lost.

In many of these cases, one is dealing with leukoagglutinin reactions and presumably this is true in case No. 2 because removal of plasma did

away with the reaction. However I would mention here the possibility of minor incompatibility involving red blood cells, not leukocytes, caused by a titre of antibodies or a type of antibody which does not give rise to overt hemolysis with hemoglobinuria. Such reactions can be related to red cell incompatibility.

It is difficult sometimes to elucidate this type of reaction. The explanation which incriminates red blood cell incompatibility can be offered in the case where one removes the buffy coat, does away with the reaction temporarily and then somewhere along the line the reactions reappear.

The third case is of interest because of the fact that monocytic leukemia is a type of leukemia which has been considered to be associated with leukoagglutinins to a much greater extent than other diseases and certainly to a greater extent than any other type of leukemia.

It may be said that once the diagnosis of monocytic leukemia is made, that person is most likely to develop leukoagglutinins, but it would seem likely here that the major reactions were due to a cold agglutinin, because warming the blood did away with the reaction. Indeed it is true that if the cold agglutinins are in high titre, warming of the blood may be necessary, but if the cold agglutinins are not of high titre it is usually sufficient to give the blood very slowly and depend on the warming that will ensue incident to very slow administration. In this case certainly one would see no contraindication to the use of warm blood. I would like to warn at this point that the warming process must be strictly supervised.

Prevention of Transfusion Reactions: R. C. Derbyshire, M.D.:

In any classification of blood transfusion reactions, it is easy to tabulate at least ten causes. More could be readily added to the list if some of the rare causes were included. The time limitation does not permit me to discuss the prevention of all reactions, hence I shall confine my remarks to general principles and discuss the prevention of some of the less common types of reactions.

The most obvious cause of transfusion reactions is the administration of the wrong type of blood. This glaring error is due to human failure at some stage of the transfusion. With modern laboratory methods, errors in actual typing and cross matching are rare. The main mistakes which are made today are clerical and involve erroneous transcription of data and mistakes in identification of patients or their blood samples. It is questionable whether or not it will ever be possible to eliminate human errors completely but it is an ideal towards which we must continually strive.

Binder, Ginsberg and Harmel[1], in studying their experiences with 81,392 transfusions at King's County Hospital, report an incidence of hemolytic reactions of between one in 2,392 and one in 4,520 with seven deaths, a mortality of one per 11,625 transfusions.

Dutra and his colleagues[2] have suggested four procedures in addition to the precautions generally observed: A pretransfusion check of the patient's and donor's blood as part of the act of bedside identification, examination of a direct smear for bacterial contamination of donor blood, special nursing care during the transfusion and sampling of the patient's blood after transfusion for evidence of hemolysis. Of major importance is the first suggestion which is worthy of detailed presentation. After the vein of the patient has been punctured, 5 ml. of blood is withdrawn and then saline but not blood is started. Five ml. of blood is then withdrawn from the bottle by puncturing the diaphragm. Rematching is carried out on a special card coated with dried collodion.

After the final check has been made the blood on the card is allowed to dry and is covered with collodion thus making a permanent record. The procedure can be carried out at the bedside. Not until after the bedside check has been made is the blood started.

It is interesting to note that in St. Vincent's as well as in many other hospitals an average of four people handle the blood from the time it is withdrawn from the donor until it is administered to the patient. At night or in cases of emergency this number can readily be increased to six or more.

For this reason it is essential that the person who finally administers the blood be one with a well developed sense of responsibility and one who is familiar with all of the precautions which must be taken. This is particularly true in cases of emergency where large amounts of blood are given over a short period of time. The sense of drama

accompanying multiple transfusions for massive hemorrhage cannot force us to relax our vigilance for one moment.

Best Method

In view of the above it is obvious that the best method for preventing reactions is not to give transfusions. But transfusions cannot be withheld when indicated. In the past two years many articles have appeared in the literature condemning the practice of giving a single pint of blood. Many writers claim that this is never indicated.

According to Moore[3], since 1917 the risk of a hemolytic reaction has decreased from one in 541 to one in 4520. The mortality rate during this period has fallen from 58 per cent to 38 per cent. But he further points out that for every known hemolytic reaction there are at least four times as many cases of sensitization to specific antibodies which may cause serious trouble during the next transfusion or the next pregnancy. The risk of an incompatible transfusion, according to this reasoning is close to one in 60.

Binder, Ginsberg and Harmel found that 53 per cent of their patients who had hemolytic reactions received transfusions for questionable reasons. They also stated that six out of seven deaths in their study were associated with transfusions of questionable necessity. Myers[4] was prompted by a recent study to caption the problem, "Transfusion by the Gill." He believes that medical staffs should seriously review the indications for transfusion and to try to reduce the number given for doubtful reasons.

In 1956 five million units of blood were transfused in 78 per cent of the hospitals in the United States[5]. If, as several writers believe, at least half were unnecessary, what an enormous waste of blood and manpower, not to mention the risk to the unhappy recipients. On the other hand it cannot be stated dogmatically that one should never administer a single pint of blood.

Nearly everyone is familiar with the patient who is in apparently mild shock, who does not respond to intravenous dextrose, dextran or other measures. Often he is then given a single pint of blood and the shock is promptly relieved. The answer to this situation may be that we are not making sufficient use of the various fractions of blood. The use of plasma fell into disrepute because of the risk of transmitting homologous serum hepatitis. We tend to forget that it has been proven that the storage of plasma at room temperature for six months will eliminate this danger. Plasma, as well as other blood fractions is an excellent adjunct in the treatment of shock and its value in this hospital, at least, has been ignored during the past ten years.

Another obvious but frequently overlooked cause of transfusion reaction is over transfusion. In recent years so much emphasis has been placed upon the quantitative replacement of blood lost at operation that many people forget that the average young adult can lose up to 15 per cent of his blood volume without serious consequences.

Measurement of Blood Loss

Le Veen, at the Clinical Congress of the American College of Surgeons in 1959 demonstrated his device for the immediate, accurate and continuous measurement of blood loss during operations. This was an important contribution but, possibly due to the high cost of the apparatus, has been insufficiently used. It is particularly valuable in long and formidable operations accompanied by great loss of blood. It eliminates guesswork, permitting adequate blood replacement and prevents the dangers of over transfusion.

Costello[6] has recently described a shock-like state due to over transfusion and reports two cases in which it occurred. The first patient died because the condition was not recognized and additional blood was given to a patient who already had been given too much.

In the second case the error was promptly recognized and the shock controlled by the withdrawal of 550 ml. of blood from the patient. It must have required courage to bleed a patient who was already in shock but it was a reasonable procedure if there was exact knowledge concerning the amount of blood lost during the operation. This is indeed an unusual sign of excessive transfusion. The usual picture is that of circulatory failure accompanied by pulmonary edema. To prevent such a reaction, particularly in a patient with cardiac disease, suspended erythrocytes should be given instead of whole blood.

Another recently recognized cause of transfusion reaction is the administration of excessive

amounts of potassium present in stored blood. Le Veen[7] found that in bank blood 40 per cent of the samples contained more than 23 m. eq. per litre of potassium, five times the physiological level. He concluded that massive blood transfusion underlay cardiac arrest in 68 out of 157 cases. He advanced the theory that the critical factor is the ratio of potassium to calcium in the venous return to the heart.

If this ratio becomes excessively high, cardiac arrest will occur. The prevention is frequent administration of calcium in massive transfusions. Schlechter, Nearon and Gibbon[8] have described an ingenious method for removing both potassium and ammonium from bank blood by passing it through an ion exchange resin column. Removal of the ammonium would of course tend to minimize reactions in patients with advanced liver damage.

Shock

In many cases of shock due to massive blood loss the administration of blood by gravity is insufficient. For this reason many transfusions are given under pressure at a rapid rate. Many special devices have been invented for this purpose. It has been pointed out that in almost all of these devices there is a real danger of air embolism and several deaths have been reported from this. This should be an exaggerated hazard in view of the fact that almost all transfusions are now administered through transparent plastic tubing through which air bubbles can easily be seen. To prevent air embolism it is essential to watch the tubing carefully and to be thoroughly familiar with the apparatus employed.

Citric acid intoxication is still another cause of transfusion reactions. A high blood citric acid concentration may occur when multiple transfusions are given to patients with liver disease or biliary obstruction. An increased blood citric acid concentration and decreased blood calcium can also occur if about 500 ml. of blood is given in 15 minutes to patients without liver disease. The ill effects are supposed to be due to hypocalcemia and the prevention is to exercise caution in administering citrated blood to patients with liver disease. Instead such patients should be given suspensions of erythrocytes.

The problem of isoimmunization should also be mentioned. According to David and Stern[9] the danger of isoimmunization increases with the number of transfusions and they designate the recipients of multiple transfusions and multiparous women as "dangerous recipients". It is estimated that one to two per cent of patients will become sensitized as the result of a transfusion. This is particularly important in women in the child bearing age. If O Rh positive blood is given in an emergency it must be remembered that 17 per cent of the white U. S. population is susceptible to sensitization to the factor which is always present in such blood. To prevent such sensitization O Rh positive blood should not be given unless its use is justified by a grave emergency.

Conclusion

In conclusion, I cannot summarize the problem of the prevention of blood transfusion reactions better than by quoting Crosby who said, "Thoughtless prescription of blood transfusion is playing Russian roulette with bottles of blood instead of a revolver; while the odds are in the physician's favor that nothing will go wrong, the patient takes the risk."

Seminar Summary: Dr. Muirhead

I think a great deal of food for thought has been offered here and I'm sure you have quite a few questions as a consequence of these talks. I would like to minimize my summary so as to allow time for discussion.

I think it is fair to say that the three cases do emphasize some prominent adversities which may follow in the wake of transfusions. The most prominent one emphasized by the first case was that of acute renal failure.

The other two cases emphasize more the problem of less severe reactions occurring in individuals requiring multiple transfusions.

In the course of considering these cases, Dr. Angle has reviewed the major causes of reaction and pointed out something about the incidence of reactions.

A major problem of transfusion reaction may be circulatory and in this connection Dr. Derbyshire has mentioned the use of concentrated red cells as a means of minimizing excess volume.

Another reaction which has been emphasized is the allergic reaction. I am reminded here of another comment in respect to the incidence of allergic reactions. A famous physician once said that if a transfusion service reports less than one per cent allergic reactions, then they are not checking their allergic reactions closely. One does expect a certain number of allergic reactions. Most of these are of a transient nature, but there is always the possibility of something more serious, such as an acute asthmatic attack, angioneurotic edema, or a anaphylaxis-like state.

Dr. Angle mentioned the causes of chill-fever reactions with emphasis on bacterial contamination, leukoagglutinins, and red cell incompatibility and pointed out that acute tubular necrosis or acute renal failure may be associated with an incompatible transfusion and hemoglobinuria, but that the primary background for this may be peripheral circulatory failure which usually accompanies the use of incompatible blood under these circumstances.

Alkalizing Agent

Certainly, one would agree that the use of an alkalizing agent is of no value here. During the war, alkalizing agents were used, such as sodium bicarbonate, in attempts to assist or prevent the kidney from undergoing injury and it was demonstrated beyond any doubt that in the first place the urine did not usually become alkaline and if it did so the patient by that time was puffed up like a balloon from salt and water retention. So this was of little value. The main point here would be to be aware of the possibility of acute renal failure, to identify it early, and to institute proper approach to management.

The first case was managed properly and recovery occurred in 18 days. Other points brought out include the factor of leukoagglutinins as a cause of reaction and it would seem that the third case represents an unusual instance where cold agglutinins were of such high titre that warming of the blood prevented the reaction. One always warns in speaking of warming blood that this should be done very carefully so as not to overheat the blood.

The last speaker covered his particular area in a very thorough manner, and I would not wish to attempt to summarize this, except to comment with respect to the percentage of unnecessary transfusions. This is something that we must always keep before us. Obviously there are unnecessary transfusions. These are not premeditatedly unnecessary transfusions always, but some of these perhaps could be so categorized.

The question of mortality of transfusions is a very difficult one, but it would follow that there is such a thing as a mortality from transfusions and one should make certain that a transfusion is indicated and be aware of the contra-indications as well as the potential complications which may result from a transfusion. In assuming this attitude, one is not assuming an anti-transfusion attitude because there is no question that the proper use of whole blood and components of whole blood has been one of the major advancements in patient care in modern times.

Discussion:

Dr. Brian Moynahan: Is there anything that one can do to treat or prevent transfusion hemosiderosis?

Dr. Muirhead: I assume that you accept the proposition that transfusion siderosis is injurious to the patient. I do accept this, but I cannot say that everyone agrees, because there are those who are uncertain that iron load is particularly injurious.

I do not know that there is anything dramatic that one can do about this problem. One point that can be made is that the intake of iron must be minimized and certainly no therapeutic iron should be given by mouth. Once the iron is in the tissue I know of no way to get it out except by phlebotomy and of course these patients are not eligible for phlebotomy.

Dr. Derbyshire: I have two questions Dr. Muirhead. I wonder if you would care to discuss your criteria for hemodialysis following transfusion reactions—how many other things do you try before you dialysize the patient, or in your institution are you very prompt to employ hemodialysis? Number two, there is something that has been troubl-

ing me for some time and that is the legal aspects and whether there have been any lawsuits resulting from dogmatic articles about one unit transfusions, and if not do you predict there will be such suits?

Dr. Muirhead: Well, I hesitate to comment on either of these questions. I have gotten away from personal experience with hemodialysis. Where I am working now it is the responsibility of other people and I am not as involved as previously. I would think however, that one would be on firm ground—and I think this is the Boston position —that hemodialysis should be individualized and that generalizations are at best only approximations and attempts to give specific criteria for general application would be somewhat risky.

One of the most prominent indications for dialysis of course, is potassium intoxication, but there are even variables with this criterion. One might give a level of say 8 meq/1 of potassium as an indication for dialysis, but even this could be erroneous in that this level may have been very slowly reached and one may wish to try the use of an exchange resin before going to dialysis; on the other hand if the potassium elevation is occurring rapidly, but is still below 8 meq/1, this would still be a reasonable indication for dialysis.

If the level goes from near normal to 8 meq/1 rapidly, you would consider dialysis more strongly than if it had gradually reached a level of 8 meq/1. Another factor here would be the degree of tissue injury associated with the rate of potassium elevation. There are other considerations in hemodialysis, and some of these include instances where the renal failure has major associated complications. Two major complications that are of major importance are, widespread traumatic tissue injury and sepsis.

Anyone who has acute renal failure associated with widespread tissue injury and in addition develops sepsis, is or becomes a candidate for hemodialysis, because the highest mortality rate is in this area. The real question is how long one should wait for hemodialysis. There are those who advocate much earlier use of hemodialysis than originally proposed; and some advocate short periods of dialysis through the course of the renal failure, particularly where there is sepsis and widespread tissue injury.

I do not know anything about legal matters and I find that every time I enter the courtroom, I know less and less, so I don't know what the answer to your second question is. I think that we have a right to become concerned over so much furor over the one pint transfusion, because we all realize that there are indications for a one pint transfusion. We mentioned such a case this afternoon, in a patient with angina who needed one pint of blood periodically to alleviate his symptoms. As a matter of fact one could argue under these conditions that two pints might be contraindicated.

Dr. Landmann: I would like to ask Dr. Muirhead how one might recognize early a transfusion reaction due to bacterially contaminated blood?

Dr. Muirhead: Before commenting on that, I would like to mention that the frequency of contamination of blood is not clearly known. There are studies which indicate a contamination rate of 2-3 per cent, but there are also studies which would indicate a contamination rate of 0.1 per cent. Fortunately whether one is dealing with a .1 per cent or a 2-3 per cent rate, these contaminants are mostly gram positive contaminants and other organisms that are relatively inconsequential as far as a reaction is concerned. They may cause a little elevation in temperature, but usually not a major chill fever reaction or shock.

Our main concern has to do with contamination by gram negative organisms which are endotoxin producers, and surely the rate of contamination by these organisms is quite small.

If one does identify a gram negative contaminant then this means a major break in the blood bank system, and I think it is right to say that if such a transfusion contaminant is identified, then the entire transfusion service should be quarantined immediately because the chances are that there will be subsequent reactions shortly, and these are of serious nature.

The most certain way to identify this type of contamination, is of course to see the organisms, and this would be by means of a smear of the blood and by culture of the blood. The reactions

which are overwhelming in this cathegory always, so far as I know, yield positive smears. By overwhelming, I mean lethal reactions. I am not aware of this being otherwise. But we can have reactions without a positive smear, but with a positive culture so that if a patient has a chill-fever reaction we do make a smear of the blood and we culture the blood.

If a patient has a chill-fever reaction and then goes into shock, particularly into the "red" type of shock—in other words if the peripheral capillaries are dilated, the skin hot and dry, and the conjunctiva extremely congested—and if the person becomes disoriented, and the blood pressure drops then we would smear the blood, culture the blood, and culture the patient.

The chance of picking up a positive blood culture in the patient is not very good and the opportunity exists only for a very short period of time, maybe only minutes, but if a positive blood culture in the patient is obtained it is a strong indication of bacterial contamination in the transfused unit.

If the smear of the blood given is positive and if the reaction is one of typical red type of shock, then we would quarantine the blood transfusion service immediately.

In regard to the patient, one would proceed to treat the patient vigorously with antibiotics and with steroid therapy. In addition to these two means, one would of course make sure that the airway is clear and that bronchial secretions do not accumulate. The use of steroids is based entirely on experimental observation. Rabbits can be partly to completely protected from endotoxin by the concommitant use of steroids, but other than this, there is no indication that steroids do something specific when used.

Dr. Edward O. Goodrich, Jr.: Would you advocate routinely smearing a sample of blood prior to transfusion?

Dr. Muirhead: At one time we encountered a series of contaminated blood involving six or seven units given between the time the first unit was given and the information came to us. We quarantined the service. Only one was fatal, fortunately.

In these cases the contaminant was in the antiseptic used on the skin. We had changed from iodine to this preparation. I doubt that we will ever change from iodine again. As a consequence of this event, our microbiology people insisted that the services must smear every bottle of blood before it be given. This simply did not work. The people who were administering the transfusions just would not do this, and we did not feel we could follow each unit to the patient to make sure that a smear was made. I think it would be a good practice if you could make it somehow practical, but how this can be done I do not know.

Dr. Goodrich: Dr. Muirhead would you tell us at what point you would begin transfusions in a patient undergoing surgery. What amount of blood loss do you feel indicates beginning replacement?

Dr. Muirhead: We try to maintain certain criteria for the use of blood when a subject has lost whole blood. There is no denying that if you lose blood and you can replace precisely the amount that was lost, homeostasis is maintained. This, of course would apply to the blood donor too. The blood donor loses a pint and insofar as anybody knows it doesn't really do very much harm to the circulation. A person can lose between a pint and two pints and be a little worse off than the blood donor because he is more susceptible to circulatory collapse, particularly under exertion.

Here the approach would be primarily one of securing rest and avoiding exertion and under these conditions a normal person may lose two pints of blood and recover without complication. It takes 36 to 48 hours or more for the patient to replenish his loss in volume, and one can hasten this with the administration of plasma.

It is after the loss of more than a liter of blood that difficulties appear, particularly after a liter and a half, and here one is dealing with acute blood loss, and one is dealing with the situation where replacement begins to be demanded. After the loss of two liters, in many people, replacement definitely is demanded. This doesn't mean that everybody will succumb to the loss of two liters of blood, but if, after the loss of a liter and a half of blood the signs of shock begin to appear—such

signs as a drop of blood pressure, elevation of pulse, cold clammy skin, etc.—this would be clear indication for transfusion, particularly if, in the recumbant position the signs of shock remain.

Now, if you don't have whole blood you can give plasma or a plasma substitute but whole blood is the fluid that is indicated. From here to more severe shock definitely whole blood is indicated. How much blood to give would depend on the clinical state of the person. I don't know of any other way of doing this, you cannot determine the blood volume that quickly. If the blood pressure goes up, sweating disappears, and the patient becomes warm, among other things these are indications that enough blood has been given.

Dr. Goodrich: My question was more, when would you start to replace blood in the patient losing blood during surgery? Would you start at 500cc or 750cc or 1000cc.

Dr. Muirhead: I would start at between 500 and 1000 ml. in a normal subject.

Dr. Angle: I have two questions. One, is there a means for correction of depression of erythropoiesis following the administration of large amounts of blood—this so called "post transfusion" anemia; two, is there any greater danger of reaction from blood used to charge an artificial kidney for dialysis of acute renal failure due to an incompatible blood transfusion than in renal failure due to other causes.

Dr. Muirhead: I would like to answer your second question first. I think you may not compound the evil, in fact you may lessen it if the titer of antibodies to begin with was such that it affected a major hemolysis with hemoglobinuria and renal failure. The primary concern relates to identification of the antibody causing the hemolysis. If this can be done with confidence and if compatible blood can be procurred with confidence, then one can proceed with confidence. I would think that this would depend primarily on the state of the antibodies that caused the reaction. Since 50 per cent of reactions are due to ABO incompatibility, the likelihood of getting around the antibodies is frequently good. I think that this would demand as much as possible knowledge of the cause of the incompatibility.

Now to the question concerning depressed erythropoiesis, this comes up particularly where one is dealing with further suppression of the bone marrow which is suppressed already, as in cases of hypoplastic marrow. It would seem that there is support for the proposition that elevation of red cell mass under circumstances where the marrow is capable of producing red cells normally will decrease red cell production, but whether this is a major contraindication of transfusions under most conditions where transfusions are indicated, I don't know.

If you feel that you should support an individual with transfusions, you raise the question of whether you are in a therapeutic paradox; does the therapy do to the subject that which you don't want done, namely to decrease the marrow output. This may be the case in chronic refractory hemolytic states, in hypoplastic states, in sickle cell anemia. From a practical standpoint I rather think that one can afford to overlook this objection because were the marrow adequate, the need for the transfusion would not have arisen.

Dr. Ellis: I would like to thank our speakers tonight for their excellent presentations and to express the appreciation of the Medical Staff to Dr. Muirhead for coming here to conduct the seminar. Next month our subject will be "Problems of Early Carcinoma of the Cervix."

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ORIGINAL ARTICLES

A Review of Infant Mortality in New Mexico and the Bordering Mexican States

(Final Section)*

ROY F. GODDARD, M.D., *Albuquerque*
STANLEY J. LELAND, M. D. *Santa Fe*
JOHN C. COBB, M. D., *Baltimore*

Research Programs in Infant Mortality

During the past eight years the Pediatric Research Department of the Lovelace Foundation for Medical Education and Research in Albuquerque, N.M., has conducted several studies pertinent to the problems of infant mortality and morbidity. Among these programs have been studies on the mechanics of breathing in the newborn,[17] the physical forces concerned with expansion of the newborn's lungs, the clinico-physiopathological relationships of broncho-pulmonary conditions in newborns and infants,[18] and studies in resuscitation, including the development of an infant hand resuscitator.[19]

Other physicians in the city have studied the effect of various aerosols on the progress of premature infants, and the influence of position on the production or formation of hyaline membrane disease.[20]

In the beginning of our studies in September 1952, one of the first steps was to organize an infant resuscitation team, clinically composed of pediatricians, obstetricians and anesthesiologists. The investigative team included in addition a roentgenologist, pathologist, respiratory physiologist, engineers, our Medical Illustration Department, otolaryngologist, neurologist, surgeons, and others which might be called upon for ancillary services.[21]

Such a team has proved itself many times over as clinically the obstetrician is able to inform the pediatrician of any difficulties expected in the delivery room and to ask him to be present at the deliveries of premature infants, difficult breach deliveries, Rh babies that he might expect trouble with, and Cesarian sections.

These two frequently then discuss with the anesthesiologist the type of anesthesia to be given.

From studies of the mechanics of breathing and studies of the physical forces concerned with expansion of the newborn lung, we were then able to proceed with the development of a small, compact, and physically and financially available, infant hand resuscitator, which closely duplicates the flow and pressure patterns of the premature and full-term infant.

The GBL (Goddard-Bennett-Lovelace) Infant Hand Resuscitator is simply a controlled mouth-to-mouth type of resuscitation, with control of the pressure, the time interval over which this pressure

*Final of three sections; published in April, May, and June 1961 issues of SOUTHWESTERN MEDICINE.

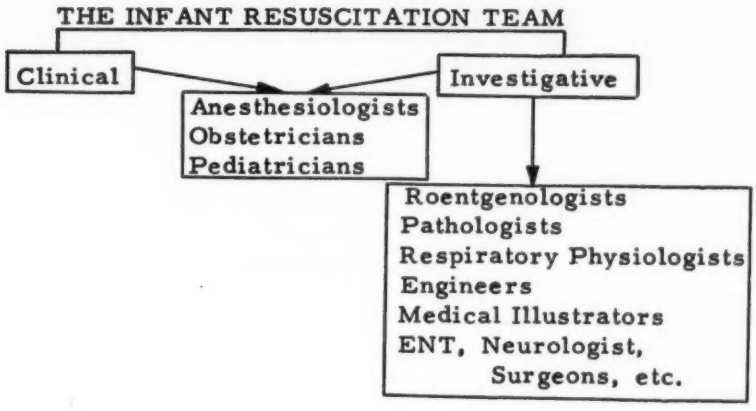

Figure 15.

is given, and the volume that is delivered. Resuscitation can be given with oxygen or with room air.

Two Maneuvers

Two simple maneuvers are required; (1) establishing a tight seal of the mask over the infant's mouth and nose, and (2) squeezing the rubber bulb to deliver the pressure set on the dial. This method of resuscitation has been fully reported on elsewhere.[19]

This resuscitator was released to various investigative hospitals throughout the country for further study. In figure 17 you see the results of a five month study done at the Los Angeles County General Hospital, in Los Angeles, California, where 5,729 infants were born during this five month period.

Two Groups

Infants were divided into a control series, which received the usual method of resuscitation, and the GBL series which received resuscitation with the Infant Hand Resuscitator. The conclusions of the study were that the use of the GBL Resuscitator showed a significant improvement in survival of premature infants weighing more than 1,000 grams; with 34 per cent dying among the controls and 13 per cent in the experimental group. Corresponding figures for full-term infants were 11 and 14 per cent respectively.[22]

Similar studies now being conducted in several hospitals in the United States and in Sweden will undoubtedly improve the infant mortality. Several meetings each year are held to discuss research on problems of the newborn, and to standardize criteria for the necessity for resuscitation and in the evaluation of infants. From such studies we expect the United States, as a whole, and many individual states and hospitals to improve their infant mortality.

Premature Nursery Center

As a collaborative effort in the State of New Mexico it is hoped that a Premature Nursery Center will be forthcoming within the near future. Such a planned center will start with an 18 incu-

Figure 16.
Clinical Use of the GBL Infant Hand Resuscitator.

STUDIES WITH GBL RESUSCITATOR
Los Angeles County General Hospital
5 Months Study Period

	Premie	Full Term	Total	% of Total
Live Births				
Control	281	2806	3087	54%
GBL Series	246	2396	2642	46%
	527 (9.2%)	5202	5729	
Neonatal Deaths	24*	17	41	0.72%
		Resuscitated Newborns		(7.2% per 1000 live births)
A. Control				
No. Resuscit.	41	82	123	
Mortality	14	9	23	19% of Resuscit.
% Mortality	34%	11%		
B. GBL Series				
No. Resuscit.	24	59	83	
Mortality	3	8	11	13% of Resuscit.
% Mortality	13%	14%		

***Includes 7 previable infants**

(Modified from Wilson and Roscoe, Calif. Med., 88: April 1958)

Figure 17.

bator capacity and will provide facilities for the hospital in which it is placed, plus facilities for infants referred by the State Health Department, the Indian Service, and other hospitals throughout the State.

Programs would include not only the actual care of premature infants, but research projects and annual sessions on premature care for the physicians and nurses of the State of New Mexico.

Summary and Recommendations

In summary, statistics have been presented on the birth rates, and neonatal mortality rates, in the United States, New Mexico, and Mexico, with comparative statistics for the State of New Mexico by County, and by races. Some of the cultural-social-economic influences in the State of New Mexico have been discussed, as have the causes of infant deaths in New Mexico.

Programs which have contributed to lowering infant mortality in New Mexico on a State-wide basis have been outlined, including the work of the New Mexico Medical Society's Maternal Infant Mortality Committee, programs of the New Mexico Public Health Department, and programs of the Division of Indian Health of the United States Public Health Service.

Existing local programs and individual hospital programs have been mentioned, together with their influence on the over-all mortality and morbidity rates. A brief mention of research studies and the part they play in this whole program has been made.

We recommend the serious consideration of the following programs.

1. Continued studies of the statistics of birth rates, neonatal mortality rates and comparative studies of countries, states, counties, and hospitals in individual cities. Efforts should be made toward more accurate analysis of the causes of death. 2. The working together of medical societies and health agencies. 3. Hospital programs, both singly and jointly. 4. Symposia and educational program, sponsored both by medical societies and various health agencies, on international, national, state and local levels. 5. Complete care, specialized when necessary, and the insistence on complete

autopsies. 6. Interest of lay groups in the problem, and encouragement of their participation in programs.

Acknowledgements

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Appendix*

1. New Mexico Medical Society Infant Mortality Study-data sheet.

2. Division of Indian Health, USPH- Phoenix branch-research sheet on Study of Fetal, Neonatal and Infant Mortality.

3. **NB-3,** Newborn Record, obstetrical and nursery data sheet, Bataan Memorial Methodist Hospital.

4. NB-4, Newborn record, premature weight chart, Bataan Memorial Methodist Hospital.

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*Charts available on request from New Mexico Department of Public Health, Maternal and Child Health Division, Santa Fe.

The Postalcoholic Syndrome

Symptomatic Control with Hydroxyzine*

HAROLD I. GOLDMAN, M.D.,** *Denver*

Confronted with an estimated five million problem drinkers[1] in this country, physicians are coming to recognize the gravity of alcoholism as a national health problem. Even if only 10 to 15 per cent of these persons are ever arrested for conspicuous drunkenness, the public cost is great. Because private physicians and hospitals have been understandably reluctant in the past to accept alcoholic patients for treatment, problem drinkers have been largely relegated for care to law enforcement agencies and public institutions. Religious groups and social agencies have also admirably assumed responsibility for managing alcoholics.

Pharmaceutical advances of the last decade have provided drugs both useful and safe for managing acute alcoholic episodes and for use in long-term treatment. As a police surgeon and in private practice, the present writer has reported[2-5] on almost two thousand patients with postalcoholic syndrome treated with various of these newer compounds. Encouraging results and reduced risks with these medicinals should greatly encourage private practitioners and institutions to accept more willingly for treatment a substantial share of alcoholic patients now in public hands.

In the treatment of the postalcoholic syndrome, the present paper will report a clinical trial and results with hydroxyzine.

Procedure and Form of the Study:

Patients were public charges, prisoners arrested for conspicuous drunkenness. All were seen in the infirmary of the City and County of Denver Jail. Single or multiple symptoms of the postalcoholic syndrome were present—nausea, tremors, hallucinations, boisterousness.

Prompt, safe control of alcoholic withdrawal symptoms was the only objective of police-medical treatment. Patients could not be released or brought before an examining magistrate until they were sufficiently controlled to comprehend the legal proceedings. If symptoms of acute delirium tremens were exhibited, patients were admitted to hospital for definitive treatment and, as such, were excluded from this clinical trial.

Included in the study were 326 patients ranging in age from 19 to 76 years. There were 212 males and 114 females. Hydroxyzine tablets were given, 200 mg. q. 4 h. If alleviation of symptoms of the postalcoholic syndrome did not occur with hydroxyzine within 16 hours (following three doses), the drug was withdrawn and considered ineffective. Prisoners were treated from 12 to 24 hours, and occasionally, somewhat longer.

*Atarax, Product of J. B. Roerig and Company, Div., Chas. Pfizer & Co., Inc.

**Police Surgeon, City and County of Denver, Colorado.

Police officers cooperated in the study by acting as observers and evaluators. These policemen, charged with thousands of alcoholic prisoners in the course of a year, are shrewd, perceptive judges of the extrinsic manifestations of the postalcoholic syndrome. A simple check sheet was prepared for yes and no answers to the following questions after each dose of hydroxyzine:

Did the prisoner's nausea improve? Did the prisoner's shakes improve? Was the prisoner able to be presented to the judge or released? Did the prisoner improve in general?

Corroboration of evaluations was possible in some cases, usually those in which the drug failed, causing the prisoner to be held over. Prisoners not given hydroxyzine, or given some other medication, served as controls. Long-standing clinical and police experience with countless such cases served as the best yardstick for evaluating the effectiveness of the drug used.

Results of the Study:

If three of the four symptoms—nausea, tremulousness, hallucinations, boisterousness and combativeness—were visibly improved or controlled within 16 hours, results with hydroxyzine were evaluated as excellent. If this did not occur, hydroxyzine was judged to be ineffective.

Accordingly, 276 of the 326 patients (85 per cent) experienced satisfactory symptomatic control; 50 patients (15 per cent) received no apparent help from the drug. These results were satisfactory in 182 of the 212 male patients (85 per cent), and unsatisfactory in 30 (14 per cent). Ninety-four of the 114 females (79 per cent) were satisfactorily controlled, while 20 (21 per cent) were not.

In most instances it was not necessary to await the time-span needed for three doses at 4-hour intervals to observe substantial improvement. Eight per cent of the males treated were controlled following one dose of hydroxyzine; 78 per cent experienced their improvement after the second or third 200 mg. dose. Among the females, 28 per cent were improved after a single dose, while 51 per cent were controlled following the second or third dose.

In no case where the results were judged satisfactory was there failure in the control of tremors. Nausea was unimproved in 28 males (15 per cent) and in 16 females (28 per cent). No instance of toxic reaction or side effect was observed or reported in this entire series of patients.

Commentary:

The physician in a police setting, treating large numbers of patients for symptoms of alcohol withdrawal, may not concern himself with the sociologic, legal, and other aspects of alcoholism. Long-term treatment or referral is not his primary medical responsibility either. The situation apropos of treating alcoholics in private practice and hospitals, however, is altogether different.

All too often, private physicians and general hospitals, lacking the inclination to intervene, shunt off elsewhere — usually to public facilities — patients who are alcoholics and in the withdrawal syndrome. Nonetheless, the physician in practice should be prepared to regard patients who are problem drinkers as legitimate medical charges who, when seen, are frequently in acute physical and psychic stress.

Much can be done to control the agitation, restlessness, and nervousness — along with the other classic symptoms — common to these patients. On many occasions, proper management will prevent immediately sending such persons back into an acute drinking episode as the only feasible escape from the urgent symptoms of alcohol withdrawal.

A compound such as hydroxyzine can be of great value to the physician undertaking to treat alcoholic patients. The drug showed itself capable of providing prompt, effective relief of most postalcoholic symptoms in the vast majority of prisoners. Continued assurance and medication with an ataractic substance will tend to allay the underlying fear and tension which are undoubtedly at the crux of most uncontrolled drinking.

If private practitioners and hospitals will continue to recognize alcoholism as a prime medical problem and more willingly provide palliation during the withdrawal syndrome with such a drug as hydroxyzine — and provide and recommend

private long-term medical and psychiatric care as needed — a certain proportion of problem drinkers will never become nuisances, prisoners, and public charges.

As is not the case with other tranquilizers, hydroxyzine has no published record of causing addiction and dependence or withdrawal symptoms. Not a single instance of toxic reaction to the drug has been reported in the literature. In addition, ingestion of high dosages daily, even for extended periods of time,[6-8] has produced no damage to blood or liver function. Successful suicide attempts with hydroxyzine have not been recorded. Where treatment of emotionally labile persons is involved, these considerations might serve to put reluctant practitioners at ease.

McGettigan[9] has reported the high effectiveness of hydroxyzine in treating patients who present themselves to private institutions for control of alcohol withdrawal and the drinking habit. He found the drug "an ideal medication for the post-alcoholic state" and that, in 85 patients treated, secondary addiction, further mood depression, convulsions, or liver damage were not encountered.

Treatment of alcoholic patients by general practitioners with hydroxyzine on an outpatient or hospital basis is recommended by Major.[10] He noted that hydroxyzine administered parenterally in 50 to 100 mg. doses brought the disturbed or combative alcoholic patient under control within 20 to 30 minutes and "may be the drug of choice for this phase of therapy."

Summary and Conclusion:

Of 326 patients treated with hydroxyzine for control of symptoms of the postalcoholic syndrome, 276 (85 per cent) were excellently controlled within 16 hours—usually after 10 hours. Dosage used was 200 mg. q. 4 h. No toxic reactions or side effects were noted. Hydroxyzine, therefore, may be unconditionally recommended to those physicians treating patients for alcohol withdrawal in a police setting.

On the basis of this experience, however, and corroborating findings of private clinicians, use of hydroxyzine may be equally urged upon private practitioners and medical-care institutions encountering alcoholic patients following an acute drinking bout. Knowledge of the fact of acute alcoholism as a pressing medical problem is essential. Proper medical management can do much to control individual problem drinkers and to prevent them from becoming public charges. The efficacy and safety of hydroxyzine seen in this study place it high on the list of agents available to all physicians undertaking medical responsibility for alcoholic patients.

1215 E. Colfax Avenue.

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Medical Art Exhibit

The 24th Annual Exhibition of the American Physicians Art Association will be held from June 26th to 30th in New York City in conjunction with the annual meeting of the American Medical Association.

The exhibit will include works of sculpture, painting, crafts and photography by physicians throughout the United States.

Further information can be obtained from Alfred A. Richman, M. D., secretary, 307 Second Avenue, New York 3, N. Y.

MEETINGS

New Mexico GPs to Meet July 17-20 in Ruidoso

The New Mexico Chapter of the American Academy of General Practice will hold its fourth annual summer clinic July 17 through July 20 in Ruidoso.

Faculty for the summer clinic will be furnished by the Texas University Medical School in Galveston. Credit for approximately 14 hours in Category I will be given physicians participating in the clinic.

Preregistration fee is $25. For further information write to Dr. R. W. Briggs, 406 North Pennsylvania Ave., Roswell, N. M.

Dr. U. S. Marshall of Roswell is president of the New Mexico Chapter of the AAGP. Other officers are Dr. Jack C. Redman, Albuquerque, president-elect; Dr. Frederick R. Brown, Roswell, vice-president; and Dr. Briggs, secretary-treasurer.

Directors are Dr. W. J. Hopkins, Lovington; Dr. H. D. Lehman, Portales; and Dr. M. A. Tanny, Albuquerque.

Delegates are Dr. Leland Evans, Las Cruces; and Dr. J. A. Rivas, Belen. Alternate delegates are Dr. A. M. Rosen, Taos, and Dr. Tanny. Dr. Evans is a member of the AAGP Commission on Education and is regional advisor for the AAGP in the Rocky Mountain states.

Dr. Redman is a member of the AAGP National Commission on Legislation and Public Policy.

COMING MEETINGS

Summer Seminar, Fundamental Aspects of Obstetrics and Gynecology, Survey of Human Genetics, University of Colorado Medical Center, Denver, June 19-21, 1961. Twelve hours AAGP Category I credit.

United States-Mexico Border Public Health Association, Annual Meeting, San Diego, June 25-29, 1961.

Rocky Mountain Cancer Conference, Hotel Brown Palace West, Denver, July 12, 13, 1961.

Summer Clinic, New Mexico Chapter, American Academy of General Practice, Ruidoso, N.M., July 17-20, 1961. Fourteen hours Category I credit.

Postgraduate Course in Pediatrics, The University of Colorado School of Medicine, Stanley Hotel, Estes Park, Colorado, August 21-25, 1961.

Western Association of Railway Surgeons, Annual Meeting, Holiday Hotel, Reno, Nev., Sept. 28-30, 1961.

Southwest Obstetrical & Gynecological Society, Eleventh Annual Meeting, Konakai Club, San Diego, Oct. 15-17, 1961.

Southwestern Medical Association, 43rd Annual Meeting, Tropicana Hotel, Las Vegas, Nev., Oct. 19-21, 1961.

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KE 3-8487 — El Paso, Texas

ROBERT J. CARDWELL, M.D.
(Diplomate American Board of Obstetrics and Gynecology)

608 University Towers Building

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